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# Health and Environmental Effects Profile for Bromoacetone

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16. ABSTRACT

The Health and Environmental Effects Profile for Bromoacetone was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for bromoacetone.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

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# HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR BROMOACETONE

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## DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

# PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or $q_1^*$ is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3112. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

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The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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## EXECUTIVE SUMMARY

Bromoacetone is a powerful lacrimator that is a liquid at room temperature (Windholz, 1983; Hawley, 1981). Although it was used as a chemical warfare agent (tear gas) during World War I (Magaha, 1964), no data are available to suggest it has been used as such since. Bromoacetone is apparently not produced, sold or used on a commercial scale in the United States.

Predictions about the environmental fate of bromoacetone are based on its physical properties and fate of structurally similar compounds because experimental data pertaining to the environmental fate processes were not located. Bromoacetone has an absorption maximum at a wavelength of ~300 nm (Mohler and Polya, 1936), suggesting that direct photolysis may be an important environmental process. Storage containers of bromoacetone should be kept out of sunlight (U.S. EPA, 1985a) because significant photodecomposition may occur. Bromoacetone may be susceptible to hydrolysis, but the lack of hydrolytic rate data precludes the determination of the significance of this reaction. Volatilization, adsorption and bioaccumulation are not expected to be important in the aquatic environment. The half-life of bromoacetone in a typical ambient atmosphere, with respect to hydroxyl radical reaction, is estimated to be ~27 days. Bromoacetone is expected to be leached easily in soil. Microbial degradation in either soil or water cannot be predicted without experimental data.

There are no monitoring data available for bromoacetone for either ambient water or air. Since bromoacetone is not produced or used commercially at present, its presence in the environment is likely to result from natural or inadvertent formation. Bromoacetone is formed naturally in the essential oil of the seaweed, Asparagopsis taxiformis, a popular edible

seaweed in Hawaii (Burreson et al., 1976). There is a potential for bromoacetone to be present in seawaters containing this seaweed. Brominated organic compounds can be formed inadvertently during wastewater chlorination treatment (Gould et al., 1983); whether the operating conditions of a chlorination treatment facility would induce inadvertent bromoacetone formation is unknown. Potential exposure to bromoacetone by inhalation and dermal exposure is probably greatest for laboratory workers using or synthesizing the compound.

The 96-hour $LC_{50}$ for bromoacetone in unspecified aquatic organisms is 10-100 mg/ℓ (HSDB, 1985). No other aquatic toxicity data were available.

Pertinent data regarding the pharmacokinetics, carcinogenicity, teratogenicity, reproductive effects and subchronic and chronic toxicity of bromoacetone in laboratory animals or humans could not be located in the available literature as cited in the Appendix. In the only available mutagenicity study, incubation of Escherichia coli strain Sd-4 in suspension with bromoacetone for 1 hour resulted in a dose-related increase in the rate of reverse mutations to streptomycin nondependence at ~0.05-1.0 mM (Hussain, 1984). No metabolic activation system was used and no positive, negative or solvent control data were given. Since no solvent control data were available, it was impossible to determine at which dose bromoacetone caused mutagenic response.

An inhalation concentration of 3200 mg/m³ (572 ppm) for 10 minutes is fatal to humans (Back et al., 1972). The odor threshold for bromoacetone is 500 µg/m³ (Sullivan, 1969).

The lack of pertinent carcinogenicity and toxicity data for bromoacetone precludes the derivation of a $q_1^*$, ADI, RQ and F. Bromoacetone is placed in EPA Category D, that is, because of lack of data it cannot be classified as to its carcinogenic potential to humans.

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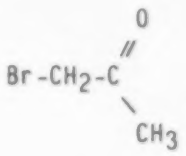
## LIST OF ABBREVIATIONS

| | |
|---|---|
| ADI | Acceptable daily intake |
| CAS | Chemical Abstract Service |
| $K_{oc}$ | Soil sorption coefficient |
| $K_{ow}$ | Octanol/water partition coefficient |
| $LC_{50}$ | Median lethal concentration |
| ppb | Parts per billion |
| ppm | Parts per million |
| RQ | Reportable quantity |
| $RV_d$ | Dose-rating value |
| $RV_e$ | Effect-rating value |
| UV | Ultraviolet |
| WS | Water solubility |

# 1. INTRODUCTION

## 1.1. STRUCTURE AND CAS NUMBER

Bromoacetone is the common chemical name, but this compound is also known as α-bromoacetone, 1-bromo-2-propanone (CAS usage), acetonyl bromide and acetylmethyl bromide (SANSS-CIS, on-line). The structure for bromoacetone is as follows:



The molecular weight for bromoacetone is 136.98, the empirical formula is $C_3H_5BrO$ and the CAS Registry number is 598-31-2.

## 1.2. PHYSICAL AND CHEMICAL PROPERTIES

Bromoacetone is a colorless liquid when pure, but it rapidly becomes violet even in the absence of air. It is soluble in acetone, alcohol, benzene and ether and is a very strong lacrimator (Windholz, 1983; Hawley, 1981). Selected physical properties are listed below:

| | | |
|---|---|---|
| Melting point: | -36.5°C | Windholz, 1983 |
| Boiling point: | 136.5°C at 725 mm Hg | Weast, 1985 |
| Specific gravity: | 1.634 (23°C) | Weast, 1985 |
| Refractive index: | 1.4697 (15°C) | Weast, 1985 |
| Log $K_{ow}$: | 0.485 (calculated) | |
| Water solubility: | $1.2 \times 10^5$ mg/ℓ (estimated)<br>$1.4 \times 10^5$ mg/ℓ (estimated) | U.S. EPA, 1985b<br>Lyman, 1985 |
| Vapor pressure<br>at 20°C:<br>at 25°C: | <br>9.0 mm Hg<br>9.4 mm Hg (estimated) | <br>Hawley, 1981 |

| | | |
|---|---|---|
| $pK_a$ (25°C) | 19.1 | Guthrie et al., 1984 |
| Vapor density: | 4.75 | Hawley, 1981 |
| Odor threshold: | 500 µg/ℓ | Sullivan, 1969 |
| Conversion factor: at 25°C | given 1 ppm = 5.60 mg/m³ | |

Some of the physical properties above were estimated because experimentally-derived values were not located. The log $K_{ow}$ of 0.485 and water solubility (at 25°C) of $1.2 \times 10^5$ mg/ℓ were estimated by the U.S. EPA's computerized GEMS model (U.S. EPA, 1985b). The water solubility of $1.4 \times 10^5$ mg/ℓ was estimated by using the log $K_{ow}$ regression equation recommended by Lyman (1985). These estimated water solubilities seem reasonable since chloroacetone, a chemically-similar compound, has a measured water solubility of $1 \times 10^5$ mg/ℓ (Hann and Jensen, 1977). Windholz (1983) reported that bromoacetone is sparingly soluble in water; however, actual solubility data were not given. The vapor pressure of 9.4 mm Hg at 25°C was estimated from an equation based on the boiling point of the liquid (Lyman, 1985). This estimation also seems reasonable because the vapor pressure of chloroacetone at 25.5°C has been measured to be 12 mm Hg (Hann and Jensen, 1977), and the vapor pressure of bromoacetone at 20°C is reported as 9.0 mm Hg (Hawley, 1981) under ambient conditions.

Bromoacetone tends to decompose; it requires an inhibitor to stabilize decomposition (Morey, 1939) and should be protected from light (Windholz, 1983). A 0.0025 M aqueous solution of bromoacetone has a measured pH value of 5.4 (Watson et al., 1935); the acidity can be attributed to coordination of the CO group with hydroxyl ion from water (Watson et al., 1935).

If bromoacetone is spilled into a body of water, it will sink to the bottom of the water column, dissolve slowly and turn violet rapidly (U.S. EPA, 1985a).

## 1.3. PRODUCTION DATA

Apparently, bromoacetone is not produced or sold commercially in the United States. Pertinent data regarding the production of bromoacetone could not be located in the available literature as cited in the Appendix.

Bromoacetone can be prepared by treating aqueous acetone with bromine and sodium chlorate at 30-40°C (Hawley, 1981) or by brominating acetone with HBr in a liquid $SO_2$ solution (Ross, 1948).

## 1.4. USE DATA

During World War I, bromoacetone was used as a chemical warfare lacrimator (Magaha, 1964), which in low concentrations induces tears and temporary eye pain. These types of warfare agents are commonly referred to as tear gases. No data were located to suggest that bromoacetone has been used as a tear gas, either for war or riot control, since World War I. Stenger (1978) and Hawley (1981) both reported that bromoacetone is used for organic synthesis, but data regarding its commercial use as an organic intermediate were not located. A potential use of bromoacetone as an intermediate is in the production of physiologically active steroid hormones (Upjohn Co., 1955).

## 1.5. SUMMARY

Bromoacetone is a powerful lacrimator that is a liquid at room temperature (Windholz, 1983; Hawley, 1981). Although it was used as a chemical warfare agent (tear gas) during World War I (Magaha, 1964), no data are available to suggest that it has been used as such since. Bromoacetone is apparently not produced, sold or used commercially in the United States.

## 2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Pertinent data regarding the environmental fate processes for bromoacetone could not be located in the available literature as cited in the Appendix. Thus, predictions of its environmental fate are based on its physical properties and chemical structure.

### 2.1. WATER

**2.1.1. Hydrolysis and Oxidation.** Although ketones are generally resistant to hydrolysis (Lyman et al., 1982), the substituted bromine group on bromoacetone may be susceptible to hydrolysis. While bromoacetone may hydrolyze in the aquatic environment, the rate of hydrolysis cannot be predicted for lack of hydrolysis data even on structurally similar compounds. Therefore, the significance of hydrolysis cannot be estimated.

Although bromoacetone is colorless when pure (Hawley, 1981), it rapidly turns violet in water (U.S. EPA, 1985a). In the absence of experimental data, however, the exact mechanism for this coloration is unclear, but it may be a result of either hydrolytic, oxidative or photooxidative mechanisms.

**2.1.2. Photolysis.** Bromoacetone strongly absorbs light in the wavelength region between 290 and 350 nm, with a UV maximum occurring at ~300 nm (Mohler and Polya, 1936). This suggests that bromoacetone may undergo direct photolysis, even in aqueous solution, but experimental data are required to predict the significance of this reaction.

**2.1.3. Microbial Degradation.** The potential for bromoacetone to be degraded by microorganisms cannot be predicted because of the lack of available data.

**2.1.4. Volatilization.** The Henry's Law constant of an organic compound can be estimated by the fragment constant methods of Hine and Mookerjee

(1975). The group fragment method yields a value of $7.4 \times 10^{-7}$ atm·m³/mol, while the bond fragment method yields a value of $3.2 \times 10^{-6}$ atm·m³/mol. These values of Henry's Law constant are indicative of slow volatilization from water (Lyman et al., 1982).

2.1.5. **Adsorption and Bioaccumulation.** The relatively low log $K_{ow}$ value and high WS of bromoacetone (see Section 1.2.) suggest that neither adsorption of bromoacetone to particulate organic matter and sediment nor bioaccumulation in aquatic organisms will be significant.

2.2. AIR

2.2.1. **Reaction with Hydroxyl Radical.** The estimated rate constant for the reaction between bromoacetone and HO· at 25°C is $0.3 \times 10^{-12}$ cm³/molecule-sec using the method reported by Atkinson (1985). Assuming an average atmospheric HO· concentration of $10^6$ molecules/cm³, the estimated half-life that is due to this reaction is 27 days.

2.2.2. **Photolysis.** The strong absorption of tropospheric sunlight by bromoacetone (Mohler and Polya, 1936) indicates that the compound has a good potential for direct photolysis in the troposphere. The fact that storage containers of bromoacetone should be kept out of sunlight (U.S. EPA, 1985a) suggests that significant photodegradation may occur; however, a quantitative estimate about the significance of this reaction in the atmosphere cannot be made because of the lack of appropriate data.

2.3. SOIL

2.3.1. **Leaching.** The $K_{oc}$ of an organic compound can be estimated from the following regression equations (Lyman et al., 1982):

$$\text{Log } K_{oc} = 3.64 - 0.55 \text{ log WS (ppm)} \qquad (2\text{-}1)$$

$$\text{Log } K_{oc} = 0.544 \text{ log } K_{ow} + 1.377 \qquad (2\text{-}2)$$

Accepting the calculated WS and log $K_{ow}$ values of $1.3 \times 10^5$ ppm and 0.485 (see Section 1.2.), $K_{oc}$ values of 7 and 44 may be calculated from Equations 2-1 and 2-2, respectively. Estimating the $K_{oc}$ from the molecular topology and quantitative structure-activity analysis method of Sabljic (1984) yields a $K_{oc}$ value of 50. Since all of the estimated $K_{oc}$ values indicate very high soil mobility, bromoacetone is expected to leach significantly in the absence of chemical or biological transformations.

2.3.2. **Volatilization.** The estimated vapor pressure of 9.4 mm Hg at 25°C indicates that bromoacetone will volatilize from dry soil surfaces. On the basis of its aquatic volatility (Section 2.1.4.), it is estimated that volatilization from moist soil will be slow.

2.3.3. **Photolysis.** Bromoacetone exposed to sunlight on soil surfaces is likely to undergo some photodegradation since it strongly absorbs sunlight. Once again, no specific data were found in the references as cited in the Appendix that would permit further quantification.

## 2.4. SUMMARY

Predictions about the environmental fate of bromoacetone are based on its physical properties and fate of structurally similar compounds since data pertaining to its environmental fate processes were not available. Bromoacetone reaches an absorption maximum at a wavelength of ~300 nm (Mohler and Polya, 1936), suggesting that direct photolysis may be an important environmental process. Storage containers of bromoacetone should be kept out of sunlight (U.S. EPA, 1985a) because significant photodecomposition may occur. Bromoacetone may be susceptible to hydrolysis, but the lack of hydrolytic rate data precludes determining the significance of this reaction. Volatilization, adsorption and bioaccumulation of bromoacetone are not expected to be important removal processes in water. The estimated

half-life of bromoacetone in a typical ambient atmosphere with respect to HO• reaction is ~27 days. Bromoacetone is expected to be easily leached in soil. Microbial degradation in either soil or water cannot be predicted without experimental data.

## 3. EXPOSURE

### 3.1. WATER

The U.S. EPA STORET Data Base contains no observations for bromoacetone. No other monitoring data were located pertaining to the detection of bromoacetone in either ambient or effluent waters.

Since bromoacetone is not produced or used commercially in the United States, its presence in water is likely to be the result of natural or inadvertent formation. Burreson et al. (1976) detected bromoacetone in the essential oil of the red alga seaweed, Asparagopsis taxiformis, which grows in the ocean around the Hawaiian Islands. Bromoacetone may be biogenetically formed in the seaweed by bromination of iodoacetone (Burreson et al., 1976). Thus, there is a potential for bromoacetone to be present in seawater containing A. taxiformis.

Gould et al. (1983) studied the formation of volatile halo-organic compounds during wastewater chlorination treatment. These authors reported that bromine, which is distributed widely in nature, is oxidized by hypochlorous acid to hypobromous acid which is capable of forming bromoorganic compounds. Upjohn Co. (1955) reported the formation of haloketones from the halogenation of enol acetates with hypohalous acid. Whether the operating conditions of a chlorination treatment facility would induce the inadvertent formation of bromoacetone is not known; however, bromoacetone has not yet been monitored in these waters, despite the potential for its formation.

### 3.2. FOOD

Burreson et al. (1976) detected bromoacetone (<1% in oil by weight) in Hawaiian seaweed. In Hawaii, this seaweed is known as limu kohu (supreme seaweed) and is highly prized for its aroma and flavor. In addition to bromoacetone, 41 other halogenated constituents were detected in this seaweed.

## 3.3. INHALATION

Monitoring data regarding the inhalation of bromoacetone could not be located in the available literature as cited in the Appendix. The potential presence of bromoacetone in seawater around Hawaii (produced by seaweed) suggests an atmospheric exposure may occur through volatilization; however, bromoacetone is not expected to volatilize significantly from water (see Section 2.1.).

The use of bromoacetone as a chemical warfare agent is obviously an intentional source for inhalation exposure; however, it has not been used for this purpose since World War I. Currently, inhalation exposure to bromoacetone is probably limited to laboratory workers using or synthesizing the compound.

## 3.4. DERMAL

Monitoring data regarding dermal exposure to bromoacetone could not be located in the available literature as cited in the Appendix. The potential for dermal exposure seems to be greatest among laboratory workers.

## 3.5. SUMMARY

No monitoring data are available for bromoacetone in either ambient water or air. Since bromoacetone is not produced or used commercially in the United States, its presence in the environment is likely to result from natural or inadvertent formation. Bromoacetone is formed naturally in the essential oil of the seaweed, A. taxiformis, a popular edible seaweed in Hawaii (Burreson et al., 1976). There is a potential for bromoacetone to be present in seawaters containing this seaweed. Brominated organic compounds can be formed inadvertently during wastewater chlorination treatment (Gould et al., 1983); whether the operating conditions of a chlorination treatment

facility would induce inadvertent bromoacetone formation is unknown. Potential exposure to bromoacetone by inhalation and dermal exposure is probably greatest for laboratory workers using or synthesizing the compound.

## 4. PHARMACOKINETICS

Pertinent data regarding the absorption, distribution, metabolism or excretion of bromoacetone could not be located in the available literature as cited in the Appendix.

# 5. EFFECTS

## 5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of bromoacetone could not be located in the available literature as cited in the Appendix.

## 5.2. MUTAGENICITY

In the only mutagenicity study available, incubation of Escherichia coli strain Sd-4 cells in suspension with bromoacetone at ~0.05-1.0 mM for 1 hour resulted in a dose-related increase in the reverse mutation rate to streptomycin nondependence (Hussain, 1984). No metabolic activation system was used and no positive, negative or solvent control data were given. Since no solvent control data were available, it was impossible to determine at which dose bromoacetone caused a mutagenic response (defined as a 2-fold increase in the number of mutants over the solvent control).

## 5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of bromoacetone could not be located in the available literature as cited in the Appendix.

## 5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of bromoacetone could not be located in the available literature as cited in the Appendix.

## 5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the chronic and subchronic toxicities of bromoacetone could not be located in the available literature as cited in the Appendix.

## 5.6. OTHER RELEVANT INFORMATION

Few additional data exist regarding bromoacetone exposure. Back et al. (1972) listed a 10-minute human inhalation lethal concentration of 572 ppm (3200 mg/m$^3$). Sullivan (1969) reported the odor threshold in humans to be

500 µg/m³. Grant (1974) reported that bromoacetone liquid was hazardous to the skin and eyes, leading to dense corneal opacity. In one case in which only a few droplets came into contact with the eye, initial opacity and necrosis were reversed after several days. Castro (1968) noted that bromoacetone did not inhibit human plasma cholinesterase in an *in vitro* titrimetric assay; more active alkylating agents, such as chloroacetophenon, were effective cholinesterase inhibitors.

## 6. AQUATIC TOXICITY

HSDB (1985) reported that the 96-hour $LC_{50}$ for bromoacetone in unspecified aquatic organisms was 10-100 mg/ℓ. Other data concerning acute or chronic toxicity, plant effects or residues of bromoacetone in aquatic biota could not be located in the available literature as cited in the Appendix.

# 7. EXISTING GUIDELINES AND STANDARDS

## 7.1. HUMAN

Pertinent guidelines and standards (e.g., U.S. EPA ambient water and air quality criteria, drinking water standards, FAO/WHO ADIs, U.S. EPA or FDA tolerances for raw agricultural commodities or foods, and ACGIH, NIOSH or OSHA occupational exposure limits) could not be located in the available literature as cited in the Appendix.

## 7.2. AQUATIC

Aquatic guidelines and standards for bromoacetone could not be located in the available literature as cited in the Appendix.

## 8. RISK ASSESSMENT

The lack of pertinent data regarding the carcinogenicity, teratogenicity, reproductive effects and subchronic and chronic toxicity of bromoacetone precludes quantitative risk assessment.

In the only available mutagenicity study, incubation of E. coli strain Sd 4 in suspension with bromoacetone at ~0.05-1.0 mM for 1 hour resulted in a dose-related increase in the reverse mutation rate to streptomycin nondependence (Hussain, 1984). No metabolic activation system was used and no positive, negative or solvent control data were given.

Back et al. (1972) reported that inhalation exposure to 3200 mg/m³ (572 ppm) for 10 minutes was lethal to humans. Sullivan (1969) reported an odor threshold for bromoacetone in humans of 500 µg/m³.

## 9. REPORTABLE QUANTITIES

### 9.1. REPORTABLE QUANTITIY (RQ) RANKING BASED ON CHRONIC TOXICITY

Pertinent data regarding the teratogenicity, reproductive effects and subchronic and chronic toxicity of bromoacetone could not be located in the available literature as cited in the Appendix. Therefore, data are not sufficient for deriving an RQ (Table 9-1).

### 9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($1/ED_{10}$) FOR CARCINOGENICITY

Pertinent data regarding the carcinogenicity of bromoacetone could not be located in the available literature as cited in the Appendix. In the only available mutagenicity study, bromoacetone treatment of E. coli strain Sd 4 at ~0.05-1.0 mM for 1 hour resulted in a dose-related increase in the reverse mutation rate to streptomycin nondependance (Hussain, 1984).

The lack of carcinogenicity data precludes the derivation of F, and bromoacetone is placed in CAG category D (U.S. EPA, 1984), that is, because of a lack of data it cannot be classified as to its carcinogenic potential for humans.

TABLE 9-1

Bromoacetone

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:

Dose:

Effect:

Reference:

$RV_d$:

$RV_e$:

Composite Score:

RQ: Data are not sufficient for deriving an RQ

## 10. REFERENCES


Atkinson, R. 1985. Kinetics and mechanisms of the gas phase reactions of the hydroxyl radical with organic compounds under atmospheric conditions. Statewide Air Pollution Research Center, Univ. California, Riverside, CA. Submitted to Chem. Rev., January 30.

Back, K.C., A.A. Thomas and J.S. MacEwan. 1972. Reclassification of materials listed as transportation health hazards. NTIS PB-214-270.

Burreson, B.J., R.E. Moore and P.P. Roller. 1976. Volatile halogen compounds in the alga Asparagopsis taxiformis (Rhodophyta). J. Agric. Food Chem. 24: 856-860.

Castro, J.A. 1968. Effects of alkylating agents on human plasma cholinesterase. The role of sulfhydryl groups in its active center. Biochem. Pharmacol. 17(2): 295-303.

Gould, J.P., R.E. Ramsey, M. Glabbai and F.G. Pohland. 1983. Formation of volatile haloorganic compounds in the chlorination of municipal landfill leachates. Environ. Impact Health Eff. 4(1): 525-539.

Grant, W.M. 1974. Toxicology of the Eye, 2nd ed. C.C. Thomas, Publisher, Springfield, IL. p. 202.

Guthrie, J., J. Cossar and A. Klym. 1984. Halogenation of acetone. J. Am. Chem. Soc. 106(5): 1351-1360.

Hann, R.W., Jr. and P.A. Jensen. 1977. Water quality characteristics of hazardous materials. NTIS-PB-285946.

Hawley, G.G. 1981. The Condensed Chemical Dictionary, 10th ed. Van Nostrand Reinhold Co., New York. p. 150.

Hine, J. and P.K. Mookerjee. 1975. The intrinsic hydrophilic character of organic compounds. Correlations in terms of structural contributions. J. Org. Chem. 40: 292-8.

HSDB (Hazardous Substances Data Bank). 1985. National Library of Medicine. U.S. Dept. Health and Human Services. National Institute of Health. On-line: October, 1985.

Hussain, S. 1984. Dose-response relationship for mutations induced in E. coli by some model compounds. Hereditas. 101(1): 57-65.

Lyman, W.J. 1985. Estimation of physical properties. (Equations 24, 40) In: Environmental Exposure from Chemicals, Vol. I. CRC Press, Inc., Boca Raton, FL. p. 31, 35.

Lyman, W.J., W.F. Reehl and D.H. Rosenblatt. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill Book Co., New York. p. 4-9, 7-4, 15-15, 15-16.

Magaha, E.P., Jr. 1964. Chemical warfare. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 4, 2nd ed. A. Standen, Ed. John Wiley and Sons, Inc., New York. p. 877.

Mohler, H. and J. Polya. 1936. Chemical warfare materials. II. Light absorption in relation to chemical constitution. Helv. Chim. Acta. 19: 1222-39. (CA 31: 1515[9])

Morey, G.H. 1939. Stabilizing halogenated ketones. U.S. Patent 2,229,625 (to Commercial Solvents Corp.) January 21, 1935. (CA 35: 2904[4])

Ross, J. 1948. Bromination of organic compounds. U.S. Patent 2,452,154 (to Colgate-Palmolive-Peet Co.). October 26, 1948. (CA 43: 2232g)

Sabljic, A. 1984. Prediction of the nature and strength of soil sorption of organic pollutants by molecular topology. J. Agric. Food Chem. 32: 243-246.

Stenger, V.A. 1978. Bromine compounds. In: Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 4, 3rd ed. John Wiley and Sons, Inc., New York. p. 256.

Sullivan, R.J. 1969. Air Pollution Aspects of Odorous Compounds. Litton Systems, Inc., Bethesda, MD. NTIS PB-188-089.

Upjohn Co. 1955. Haloketones. British Patent 732,577. June 29, 1955. (CA 50: 7154i)

U.S. EPA. 1984. Guidelines for Carcinogen Risk Assessment. Federal Register. 49(227): 46294-46301.

U.S. EPA. 1985a. OHM-TADS (Oil and Hazardous Materials Technical Assistance Data System). on-line: December 11, 1985.

U.S. EPA. 1985b. U.S. EPA Computer printout: Graphic Exposure Modeling System (GEMS) EXAMS model. Office of Toxic Substances, Washington, DC.

Watson, H.B., W.S. Nathan and L.L. Laurie. 1935. Acidities of ketones and the mechanism of prototropic change. J. Chem. Physics. 3: 170-4. (CA 29: 28253)

Weast, R.C., Ed. 1985. CRC Handbook of Chemistry and Physics, 66th ed. CRC Press, Inc., Boca Raton, FL. p. C-51.

Windholz, M., Ed. 1983. The Merck Index, 10 ed. Merck & Co., Inc., Rahway, NJ. p. 193.

## APPENDIX

## LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN Internation
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in October, 1985. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation through 1984). Cincinnati, OH. 486 p.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1985-1986. Cincinnati, OH. 114 p.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2A. John Wiley and Sons, NY. p. 2878.

Clayton, G.D. and F.E. Clayton, Ed. 1981. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2B. John Wiley and Sons, NY. p. 2879-3816.

Clayton, G.D. and F.E. Clayton, Ed. 1982. Patty's Industrial Hygiene and Toxicology, 3rd rev. ed., Vol. 2C. John Wiley and Sons, NY. p. 3817-5112.

Grayson, M. and D. Eckroth, Ed. 1978-1984. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 24 Vol. and Supplement.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Littleton, MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. IARC, WHO, Lyons, France.

ITII (International Technology Information Institute). 1982. Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal with Toxicity and Hazard Data. ITII, Tokyo, Japan. 700 p.

NTP (National Toxicology Program). 1985. Toxicology Research and Testing Program. Chemicals on Standard Protocol. Management Status.

Ouellette, R.P. and J.A. King. 1977. Chemical Week Pesticide Register. McGraw-Hill Book Co., NY.

Sax, I.N. 1984. Dangerous Properties of Industrial Materials, 6th ed. Van Nostrand Reinhold Co., NY.

SRI (Stanford Research Institute). 1985. Directory of Chemical Producers. Menlo Park, CA.

U.S. EPA. 1985. Status Report on Rebuttable Presumption Against Registration (RPAR) or Special Review Process. Registration Standards and the Data Call in Programs. Office of Pesticide Programs, Washington, DC.

U.S. EPA. 1985. CSB Existing Chemical Assessment Tracking System. Name and CAS Number Ordered Indexes. Office of Toxic Substances, Washington, DC.

USITC (U.S. International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982, USITC Publ. 1422. Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book, Vol. 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Service Resource Publ. 137, Washington, DC.

McKee, J.E. and H.W. Wolf. 1963. Water Quality Criteria, 2nd ed. Prepared for the Resources Agency of California. State Water Quality Control Board. Publ. No. 3-A.

Pimental, D. 1971. Ecological Effect of Pesticides on Non-Target Species. PB-269605. Prepared for the U.S. EPA, Washington, DC.

Schneider, B.A. 1979. Toxicology Handbook. Mammalian and Aquatic Data. Book 1: Toxicology Data. Office of Pesticide Programs, U.S. EPA, Washington, DC. EPA 540/9-79-003. NTIS PB 80-196876.